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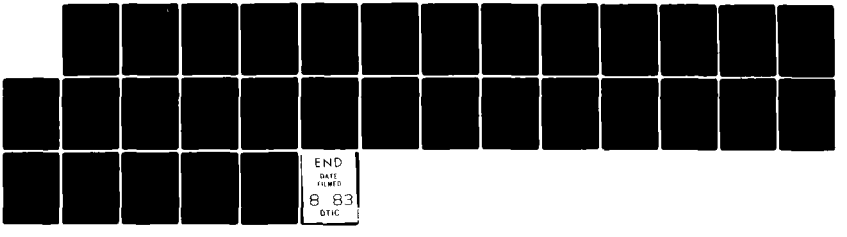

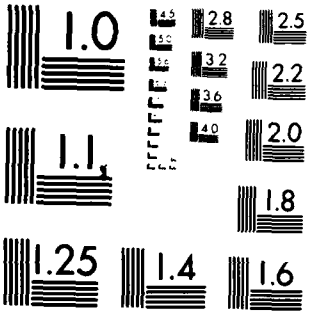

THE MECHANISM OF BLOOD FUNCTION AND PRODUCTION AFTER INJURY

ANNUAL PROGRESS REPORT

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EFFECTS OF NUTRITIONAL ROUTE ON SURVIVAL OF MALNOURISHED AND WELL NOURISHED RATS AFTER SEPTIC CHALLENGE AND ON THE BODY COMPOSITION OF PREVIOUSLY MALNOURISHED ANIMALS

Kenneth A. Kudsk, MD and George F. Sheldon, MD

Intravenous nutrition is an effective alternative to gastrointestinal feeding in patients with anatomic or functional loss of gastrointestinal integrity. In these patients, total parenteral nutrition (TPN) can provide the nitrogen, calories, vitamins, minerals and electrolytes necessary to generate positive nitrogen balance with resulting maintenance or increase in body cell mass without edema formation. Intravenous feeding, however, bypasses the complex processing of foodstuffs which normally occur in the G-I tract and some of the protein-calorie malnutrition (PCM) associated morphological[1, 2] and functional[3, 4] defects of the gastrointestinal tract persist after IV feeding.[5] Frequently, no absolute contraindication to enteral feeding exists, however, and technical developments in enteric feeding systems and chemically defined diets allow physicians the opportunity to infuse nutrients enterally despite anorexia or varying degrees of malabsorption. The recognition that postoperative ileus frequently spares the small intestine[6] has made percutaneous placement of needle jejunostomies[7] a practical access route in patients who might otherwise receive nutrients intravenously. Since both routes are feasible in such a population, selection of nutritional approaches should be predicated on the basis of preservation of important visceral protein function such as host defense.

Increased susceptibility to infection commonly occurs in association with PCM[8]. Our previous experiments have demonstrated the effects of PCM on mortality with experiments utilizing hemoglobin E.Coli peritonitis.[9, 10] Well-nourished rats refed with standard diets withstood intraperitoneal administration of hemoglobin-E.Coli better than malnourished animals. Although intravenous feeding of the same solution

into malnourished rats resulted in weight gain and positive nitrogen balance, the mortality rate from a septic challenge between the malnourished and intravenously fed cohorts was similar. This experiment did not determine whether the lack of improvement with IV feeding was due to the route of nutritional repletion, or to the absence of some substrate within the TPN solution. Subsequent experiments have shown that this does not appear to be substrate related.

This study investigates three aspects of enteral versus parenteral feeding: the role of route of nutrient administration on survival after septic challenge of previously (a) malnourished; (b) well-nourished animals; and (c) on the final body composition of malnourished rats repleted via the two routes.

Material and Methods

Experiment 1.

Effect of route of administration on survival of malnourished animals repleted intravenously or enterally.

Fifty-two female Fischer rats (120-160 grams) were randomized into 1 of 3 experimental groups. For the first 14 days of the protocol, all animals were fed a protein-free, 2% agar diet (U.S.P. XV-ICN Nutritional Biochemicals) containing adequate vitamins and minerals and some carbohydrate designed to produce PCM. The 16 animals selected for eventual oral repletion with TPN solution (ENT group) were started into the protocol 24 hours prior to the remaining 36 animals (Fig. 1). After 10 days of eating the 2% agar diet, each animal was lightly anesthetized with Metafane, weighed, and the external jugular vein cannulated with a silastic catheter through a neck incision to provide a route for infusion of nutrients. The catheter tip was advanced into the right atrium of the heart, secured within the vein by silk suture, and tunneled subcutaneously to the back of the neck. The catheter was brought through a midline incision and protected by a flexible steel sheath, the base of which was securely fastened to the musculature of the neck. This model provides

the capability of unrestrained mobility in the metabolic cages while solutions are infused. Normal saline solutions were utilized to maintain catheter patency until nutrient regimens were begun.

All animals were catheterized, insuring equal invasive management of the rats, whether fed by the IV or oral route. On the 14th day, the agar diet was discontinued and 16 animals (ENT) were fed ad lib a 25% dextrose-4.25% amino acid (Freamine II) solution containing vitamins and electrolytes (Table I). After 24 hours, and on each subsequent day, the volume ingested by each ENT animal was measured and an equal volume of solution (corrected for weight differences) was infused into a pair-fed rat over the subsequent 24 hours (IV group). The remaining 20 PCM rats were provided unlimited access to standard rat chow (RC), to return to a state of normal nutrition (RC group); one anidmal in this group died during the course of the experiment. RC and ENT animals were given a constant infusion of normal saline to maintain catheter patency during the refeeding period. All animals had unlimited access to water.

During the refeeding phase, urine and feces were collected daily and analyzed for nitrogen excretion suing the micro-Kjeldahl technique. After 13 days of ENT refeeding (12 days for IV and RC rats), the TPN and rat chow refeeding was discontinued and all animals were fasted for 24 hours, while catheter patency was maintained by infusion of normal saline.

E. Coli-hemoglobin adjuvant solution was produced from a stock culture of E.Coli previously obtained from the laboratory of Dr. L.B. Hinshaw. The E.Coli suspension was adjusted with normal saline to a predetermined density using a Zeiss spectrophotometer. Blood was harvested from donor cohort female Fischer rats, washed, and combined with the E.Coli to make a 4% hemoglobin mixture with 10⁹ organism/ml of E.Coli. Bacterial counts in the inocula were confirmed by serial plate dilution and by counting with a colony counter.

Following the 24 hour pre-challenge fast, the rats were anesthetized, weighed,

injected intraperitoneally with 0.7 ml/100 grams weight of the E.Coli-Hb mixture, and returned to their metabolic cages.

All animals surviving the challenge were sacrificed at 3 days. The carcasses of 8 ENT and 8 IV rats were dried to a constant weight after removal of the intestines, so that total body nitrogen (TBN) could be measured using the micro-Kjeldahl technique. Body composition and balance studies were performed to insure comparable nutritional repletion.

Statistical analysis of survival was determined by the log-likelihood ratio. Statistical comparisons between ENT, IV and RC rats were performed using analysis of variance and Neuman-Keuls test for parametric data. Statistical comparisons of body composition between ENT and IV pairs were performed using Student's test for paired data. All values were indexed to weight and used body weight after protein depletion as the indexing weight, with results expressed as mean (X) and standard error of the mean (SEM).

Experiment 2.

Effect of route of nutrient administration on the final body composition of malnourished animals after repletion.

The protocol above was repeated to obtain body composition data. Twenty-four female Fischer rats were weighed and divided into groups of 3 animals with similar body weights. Randomly, one animal from each group destined for enteral repletion (ENT-COMP) was given ad lib access to 2% agar diet 24 hours prior to the remaining 16 animals. On the 10th day of agar diet, 16 animals had intravenous cannulation (ENT-COMP and IV-COMP animals). Gastrostomies were placed into the final 8 animals (GAST-COMP). On the 12 day of repletion, all animals were sacrificed.

The stomach, small intestine and colon were carefully divided at their mesenteries and removed from the carcasses with separation of the individual organs at the pylorus and ileocecal valve. The small intestine was divided at its midpoint. Each luminal organ was opened along its length, irrigated to remove contents, blotted and weighed. The liver was removed at its vascular attachments, allowed to drain its blood, blotted and weighed. The carcass, liver and small intestine were then dessicated and total nitorgen measured with Kjeldahl technique. Samples of the carcass were weighed before and after ether extraction and total lipid content calculated. Daily urine collections were also analyzed by Kjeldahl technique to allow nitrogen balance studies. All results are expressed as mean + SE. Statistical analysis was carried out by analysis of variance and Newman-Keul's test. One GAST animal died in the early postoperative period.

Experiment 3

Effect of route of administration on survival of normally nourished rats.

Female Fischer well nourished rats were paired by weight. One animal from each pair had placement of an intravenous catheter while the other had gastrostomy placement and both were given ad lib access to normal rat chow for the first 3 postoperative days. On the 4th day, each animal had infusion of standard 25% dextrose-4.25% Freamine solution at 32 ml/100 gm weight. Infusions were stopped on the 12 day and the animals were fasted 24 hours prior to the septic challenge. Daily urine collections were obtained and nitrogen balances calculated. This protocol is still under investigation at the time of this writing. Statistical analysis was carried out by unpaired Students t-test and one log-likelihood ratio.

Results

Experiment 1. Rats were fed almost identical volumes of TPN solution orally or intravenously, daily, which resulted in equal nitrogen balance of the 2 groups (Table II). In spite of equal substrate administration, IV rats gained significantly more weight than

ENT fed rats ($p < .001$) (Table III). Rats fed a regular diet (RC) had a greater nitrogen intake than ENT or IV rats. However, because of large variations, nitrogen balance was not significantly better. Weight gain in RC rats was significantly greater than ENT rats but not IV animals. When indexed to initial weight, IV and RC rats gained significantly more weight per day than ENT rats (Fig. 2, Table III).

To evaluate the greater weight gain in the IV group as compared to the ENT group, total body water (TBW) and TBN were performed on carcasses of 8 ENT-IV pairs (Table IV). IV animals had significantly lower TBW ($p < .01$) than their pair-fed (ENT) counterparts. TBW measurements of normally fed rats is approximately 70%, which is comparable to ENT animals. Although no carcass fat analysis was performed, it was suspected that the greater weight gain of the IV rats was due to fat deposition. Experiment 2 investigated this assumption.

Mortality following E.Coli-Hb peritonitis was used to evaluate host resistence following oral or IV repletion of the malnourished rats. Survival was unrelated to the type of oral nutrition provided; both rat chow and TPN solution taken orally improved survival. Rats fed intravenously (IV) had significantly greater mortality at 24 hours after peritonitis than orally (ENT or RC) refed rats ($p < .05$). Although these differences were not significant at 48 hours (ENT vs. IV, $p = .08$), this was probably due to the population size. ENT and RC surviving animals appeared to recover from peritonitis sooner than IV refed survivors.

Experiment 2

Body and organ weights

There was no significant difference between groups in the amount of TPN solution (and therefore, nitrogen or calories) administered to the 3 groups during the course of the experiment (Table V). Although starting weights were essentially equal among the groups, IV-COMP animals gained significantly more weight ($p. < .05$) with greater final

body weights than GAST-COMP or ENT-COMP animals, confirming the results of Experiment 1.

The weight of the proximal small intestine was significantly greater in the ENT-COMP ($p < .001$) and GAST-COMP ($p < .001$) animals than in IV repleted rats (Table VI). Surprisingly, weights of proximal small bowel in ENT-COMP animals were also greater than in the continuously fed GAST-COMP rats ($p < .005$). These relationships did not apply to the distal small bowel, however, where weights were significantly greater in the ENT-COMP rats than either GAST-COMP or IV-COMP animals ($p < .025$). No statistical difference existed between the latter groups. Stomach weights were greater in both GAST-COMP and ENT-COMP animals than IV-COMP rats ($p < .05$), however. Colon and liver weights were similar in all groups.

Nitrogen distribution (Table VI)

Although nitrogen administration was similar in all groups, nitrogen balance was better in the ENT-COMP group ($p < .025$) than either GAST-COMP or IV-COMP animals. Total body nitrogen did not reflect this difference, probably because of the relatively small contribution of this additional nitrogen to total carcass nitrogen.

Organ nitrogen distribution was significantly affected by route of administration, however. The analysis of small bowel nitrogen paralleled weight data. IV-COMP animals had the lowest proximal intestinal nitrogen values which were significantly lower than either orally fed groups ($p < .05$). The proximal intestine of discontinuously fed ENT-COMP animals contained more nitrogen than GAST-COMP rats ($p < .05$). Distal nitrogen was not preserved in either GAST-COMP or IV-COMP animals which were significantly lower than ENT-COMP animals ($p < .05$). These results remain consistent, even when nitrogen data is indexed against weight or total body nitrogen.

Liver nitrogen appeared to be maintained less in the IV-COMP animals. Although no statistical difference exists between groups when all animals are analyzed, one IV-COMP rat had a much larger liver than any other animal studied (Fig. 2). If this single

animal is discarded from the analysis, the livers of IV-COMP rats have statistically less total nitrogen than either orally fed group ($p < .05$).

Fat analysis (Table VII)

The IV-COMP animals appeared to be fatter than ENT-COMP or GAST animals during autopsy with an increased deposition in the subcutaneous space and mesentery. Lipid extraction confirmed this impression, as total carcass lipid, lipid/loo gram final weight, and lipid/total body nitrogen were significantly greater in IV-COMP animals than GAST-COMP or ENT-COMP rats, confirming the impressions from Experiment 1.

Experiment 3

There was no significant difference between the volume of TPN solution administered to animals fed intravenously or via gastrostomy (Table VIII). Previously well nourished (WN) GAST animals appeared to have identical weight gains to IV-WN animals. Similar weight gains in normally nourished animals fed enterally and intravenously is consistent with the findings of others. There was no difference in nitrogen balance between the two groups. At the time of this manuscript's preparation, no statistically significant differences exist between IV and GAST animals, although it is obvious that the 2 groups are diverging and that with increased population size, a confidence level of 95% will be reached (Table IX). GAST animal results most closely paralled results in control rats. Again, most of the differences occur within the first 24 hours, suggesting some inadequacy in response to the challenge in IV fed animals. It appears that both state of nutritional status and route of nutritional support are important factors to survival after the challenge, and that the factors may be additive. Since greater numbers of well nourished animals are required to show the effects of route of administration on survival, it seems that prior body state and route are additive factors in this model of sepsis. Those animals sustaining prior protein depletion in addition to the effects of route of feeding (Experiment 1) demonstrate these effects most acutely.

Discussion

Previous experiments from this laboratory using a PCM model challenged by E.Coli-Hb peritonitis suggest that nutritional state influences survival[9, 10]. Normally fed rats and PCM rats fed standard rat chow 2 weeks after a protein depletion diet have a 50% to 65% survival rate 48 hours after the septic challenge. Rats with 14 days of protein depletion have a survival rate of less than 20%. Oral ingestion of TPN solution in quantities isocaloric and isonitrogenous with intravenous solution is associated with superior survival than if the same substrate is given intravenously. The reason for improved survival after oral refeeding is unclear. The rapid death rate during the first 24 hours in rats fed intravenously suggests a metabolic or immunologic defect. It is possible that immunologic or metabolic factor is neither maintained nor corrected by intravenous administration of nutrients.

Several authors have studied models of septic peritonitis and proposed mechanisms of death.[11] Mortality rate clearly correlates with reduced peritoneal clearance of bacteria[12, 13] documented by colloidal-protein absorption studies and by recovery of intraperitoneal bacteria. Continued intraperitoneal localization of bacteria results in increasing concentrations of bacteria after growth and multiplication. Proliferation of bacteria may result in release of greater amounts of toxic products being absorbed, or greater blood stream invasion by live bacteria. Sterile toxic products which accumulate in peritoneal fluid following ligation of the appendix in dogs increase bacterial virulence.[14] The addition of hemoglobin to bacteria in this peritonitis model may impair local or systemic defense mechanisms.[15] It does appear that PCM influences the function of the reticulo-endothelial system to sepsis[16] but whether IV feeding exerts its deleterious effects in this manner is unknown.

The different weight gains between the ENT and IV groups fed equal substrate associated with identical nitrogen balance and total body nitrogen values is of interest.

Bury et al[17] noted greater weight gain in orally fed rats than in IV fed normal rats. When Johnson et al[11] studied the effects of oral vs. IV feeding on the G-I tract, they noted that 5 harnessed rats taking 49K-cal per day of solution failed to gain weight after 8 days of oral intake, while IV fed rats receiving the same amount of IV fluid gained weight through the course of the experiment. The role of the G-I tract in malnutrition and refeeding is clearly important. Considerable attention has been devoted to the atrophy of the villous structures of the G-I tract[2, 5, 18, 19] and the return of enzymatic function[18] as malnutrition is corrected. Moreover, the essential role of intraluminal foodstuffs in return of atrophied intestinal villi to normal absorptive capacity[19] following a period of exclusion or malnutrition is well established. While Bury's data support the proper concept that gastrointestinal feeding is superior to parenteral feeding, the present study demonstrates that weight gain is not necessarily an appropriate index of nutritional recovery and that prior state of nutrition as well as route of administration influences utilization of substrates and subsequently body composition dramatically. In short, visceral protein recovery occurs even without great weight gains.

Although the present study demonstrates the importance of oral intake, and possibly intermittent oral intake, on the maintenance and maximal recovery of visceral protein, the mechanisms for this are not well defined. The gastrointestinal tract contains active tissues undergoing rapid protein synthesis, rapid turnover and a high metabolic rate. Developmentally, it seems appropriate that the body should reduce the mass of this metabolic drain when food is scarce. Within 24 hours of starvation[19] significant decreases in the mass of the rat's small intestine can be measured so that by 6 days[2] the weight loss of the small intestine is out of proportion to losses from the rest of the body. Intravenous feeding of glucose-amino acid solutions does not obviate these changes.[5] During starvation of rats, visceral protein may meet as much as 40% of the body's metabolic demands through gluconeogenesis.[21] Somehow, ingestion of food maintains these tissues. Decreases in enzyme activity[5, 19, 22, 23] pancreatic acinar cells,[3,4] and gastric secretion[25] occur over similar time courses. These changes do

in humans, albeit at a slower rate.[18]

Since the small intestine may release its labile proteins rapidly during early starvation, it follows that when foodstuffs are presented to the protein-depleted intestine, the intestinal mucosaa demonstate an avidity for protein not seen in well nourished animals. When protein depleted rats were given labelled amino acids intragastrically, the depleted intestine demonstrated greater specific activity for the amino acid than well fed controls.[26] The proximal intestine, which demonstrates the greatest decrease in mass during starvation,[5] seems to show the greatest avidity for the amino acids during oral refeeding. This probably accounts for the rapid recovery of bowel mass after refeeding.

Parenteral nutrition does have positive effects on visceral proteins, such as albumin and liver nitrogen in malnourished animals,[27o] but there is little doubt that enteric administration generates a much different response to the nutrients which influences not only the local visceral composition but the composition of peripheral tissues as well. Brooke and Ashworthe[28] documented 20-25% increases in the metabolic rate of malnourished infants after feeding during their rapid phase of recovery: these increases substantially decreased after recovery to more normal levels. The increases in metabolic rate appear to reflect protein synthesis[29] and may be due, at least in part, to recovery of the metabolically active intestine.

An advantage to enteral feeding certainly resides in the usual metabolic processing performed by the splanchnic bed. Processing by the mucosa and liver influence both portal and systemic amino acid profiles.[30] Glucose administered enterally into patients stimulates a much greater release of insulin than the same dose given intravenously[31] -- implying that intestinal factors are important in the generation of normal food processing.[32] Glucose removal after oral loading progressed at a rate 3 times faster than after IV administration[33], but only 30-40% of the oral glucose load escapes the splanchnic bed actuely in normal individuals. Human given a 100 gram glucose load

release only 15% from the splanchnic bed for disposal by peripheral tissues.[36] The remaining 85 grams either remain within the splanchnic system or are released to satisfy obligatory demands of glucose requiring tissues. Parenteral administration delivers the bulk of the glucose load to the periphery and the splanchnic circulation sees only that part delivered through the arterial circulation. In comparison to the malnourished infants fed orally, (sited above) protein depleted patients given glucose based TPN solution had no increase in oxygen consumption, but did have documented lipogenesis.[35] Others[36] have also noted significant increases of body fat in parenterally fed patients.

The results of our study demonstrated that enteral administration of nutrients recovers and maintains visceral proteins significantly better than the same nutrients given intravenously. Weight gain cannot be used as a comparative indication of increases in lean tissue between malnourished patients fed enterally, and those fed intravenously. The increased metabolic response associated with recovery during enteral repletion and the splanchnic manipulation of foodstuffs may account for the lesser peripheral deposition of fat in enterally fed animals. Because metabolic rates and nutrient fluxes were not measured, these hypotheses remain speculative.

Conclusion

These experiments depict the importance of enteral administration of nutrients whenever feasible. Enteral feeding improved resistance to peritonitis in malnourished animals and maintained resistance in previously well nourished rats (recognizing that at the time of manuscript preparation, statistical significance was not yet obtained in the latter group). When assaying nutritional support and its effect upon body composition, one must consider prior state of the body composition as well as nutritional route. Visceral protein appear to be repleted and maintained in malnourished animals with enteral feeding at the expense of fat deposition.

TABLE I

NUTRIENT SOLUTION

Dextrose 25%

Freamine II 4.25%

NaCl 25 mEq/L

K_3PO_4 20 mEq/L

$MgSO_4$ 16 mEq/L

Ca. Gluconate 2.7 mEq/L

K Cl 4.0 mEq/L

MVI 1 ampule/Liter

TABLE II

EXPERIMENT I - PRETREATMENT: MALNOURISHED

	ENT	IV	RC
TPN Administered (ml/100 gm/day)	31.6±1.4	31.9±1.4	--
Nin(mg)/100 gm/day	197.8±8.8	199.8±8.6	334.0±9.8*
N.Balance (mg)/day	62.8±3.0	62.5±5.5	78.6±7.9

($\bar{X}$ - SEM)

* Versus ENT, IV $p < .001$.

ENT and IV animals were given equal volumes of TPN solution. RC animals ingested significantly greater amounts of nitrogen, but nitrogen balance was not significantly improved.

TABLE III

EXPERIMENT 1: PRETREATMENT: MALNOURISHED

	ENT	IV	RC
Weight after Agar	107.3 ± 2.4	115.1 ± 3.1	118.3 ± 2.6
Weight after Re-feeding	109.2 ± 3	$127.2 \pm 4.3^{**}$	$132.4 \pm 2.5^{**}$
Weight change/day	$.14 \pm .23$	$.94 \pm .17^{**}$	$1.1 \pm .11^{**}$
Weight change/d/100/gm	$.17 \pm .24$	$.81 \pm .15^{\S}$	$.98 \pm .20^{**}$

($\bar{X} \pm$ SEM)

* Versus ENT $p < .05$

** Versus ENT $p < .01$

§ Versus ENT $p < .025$

TABLE IV

BODY COMPOSITION - EXPERIMENT 1

	ENT	IV	
Total Body Nitrogen (gm)/ 100 gm	3.1±.1	3.05±.1	NS
Total Body Water (%)	69.8-.6%	64.9-1.4%	p .01

($\bar{X}$ ± SEM)

IV fed animals have equal total body nitrogen, but less total body water than ENT fed animals.

TABLE V

BODY COMPOSITION - EXPERIMENT 2

	ENT-COMP(8)	GAST-COMP(7)	IV-COMP(8)
WEIGHT AFTER AGAR	105±2	106±2	107±5
WEIGHT AFTER REFEEDING	114±1.9*	115.7±2.2*	130.8±6.6
WEIGHT CHANGE	8.7±1.4*	9.9±1.8*	21.3±5.4
TPN given/100 g	398.5±7.8	409.4±10.7	409±9.3
Nit. Bal (mg/100 g)	.99±.04	.82±.03**	.83±.04**

($\bar{X}$ ± SEM)

* vs IV $p < .05$

** vs ENT $p < .025$

TABLE VI

ORGAN ANALYSIS -- EXPERIMENT 2

WEIGHT (gms)	ENT-COMP(8)	GAST-COMP(7)	IV-COMP(8)
LIVER	$4.978 \pm .164$	$4.52 \pm .345$	$5.20 \pm .58$
PROXIMAL SMALL BOWEL	$1.61 \pm .05$*	$1.40 \pm .06$*§	$1.08 \pm .06$
DISTAL SMALL BOWEL	$1.003 \pm .04$	$.857 \pm .03$§§	$.837 \pm .05$§§
Nitrogen (gms)			
LIVER	$.131 \pm .005$	$.121 \pm .004$	$.112 \pm .007$
PROXIMAL SMALL BOWEL	$.042 \pm .001$	$.037 \pm .002$§	$.028 \pm .001$**§
DISTAL SMALL BOWEL	$.028 \pm .002$	$.023 \pm .001$§	$.022 \pm .001$§
TOTAL BODY	$3.51 \pm .03$	$3.45 \pm .07$	$3.69 \pm .13$

($\bar{X} \pm$ SEM)

* vs IV $p < .001$

** vs GAST $p < .05$

§ vs ENT $p < .05$

§§ vs ENT $p < .025$

TABLE VII

BODY LIPID ANALYSIS - EXPERIMENT 2

	ENT-COMP(7)	GAST-COMP(7)	IV-COMP(8)
TOTAL CARCASS LIPID	6.15±.84	7.93±.73	12.58±2.02*
LIPID/100 gm WEIGHT	5.40±.69	6.84±.57	9.51±1.18*
LIPID/TBN	1.76±.25	2.30±.20	3.38±.444*

($\bar{X}$ - SEM)

* vs. ENT, GAST, $p < .05$

TABLE VIII

PRETREATMENT -- WELL NOURISHED ANIMALS - EXPERIMENT 3

	GASTROSTOMY	INTRAVENOUS
INITIAL WEIGHT	149.2 ± 1.78	150.6 ± 1.8
FINAL WIEGHT	152.9 ± 1.7	154.2 ± 2.1
WEIGHT Δ/100g/d	$.21 \pm .05$	$.20 \pm .05$
TPN GIVEN/100 g	404.9 ± 8.3	395.5 ± 8.9
NITROGEN BAL. (mg/d)	74.8 ± 4.3	71.2 ± 3.3

($\bar{X} \pm$ SEM)

TABLE IX

SURVIVAL OF WELL NOURISHED ANIMALS ON:

	DAY 1	DAY 2
GAST (n= 28)	53.5%	45.7%
IV (n= 30)	29.7%	23.1%
CONTROL (n= 20)	55.0%	40.0%

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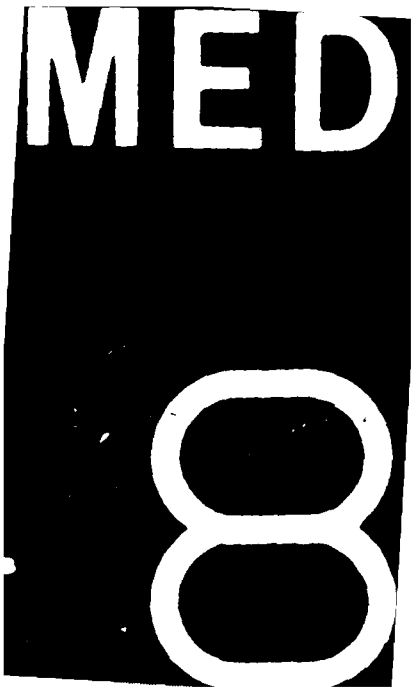
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